Cardiovascular Risk in Obesity what is more? The Omentin

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Abstract

Obesity increases the development of morbidity and mortality via varieties of severe cardiovascular diseases. Omentin is a newly identified adipokine selectively expressed in visceral adipose tissue. Clinical and experimental investigations suggested omentin as a critical mediator of cardiovascular homeostasis. Its ability to decrease insulin resistance in combination with its anti-atherogenic and anti-inflammatory properties makes it a promising therapeutic target. Much work remains to be done to understand the fascinating biology of omentin as a diagnostic and prognostic biomarker which will likely benefit the obese individuals with or without heart diseases.

Keywords: Obesity; Cardiovascular diseases; Omentin

Currently, obesity is one of the most commonly public international problems that decrease the life expectancy. It increases developing morbidity and mortality via varieties of severe cardiovascular diseases. Adipose tissue is not only energy storage, but also the largest active endocrine organ in the body by releasing large number of bioactive mediators (adipokines). The adipokine secretion is significantly changed towards a diabetogenic, pro-inflammatory, and atherogenic pattern [1].

Omentin is a newly identified adipokine selectively expressed in visceral adipose tissue in two omentin genes, Omentin -1 and Omentin -2. In contrast to other types of adipokines, low omentin expression was observed in patients with obese complications. The discovery of omentin is now exciting in several cardiovascular researches. Clinical and experimental investigations performed both in vivo and in vitro have suggested omentin as a critical mediator of cardiovascular homeostasis. It involved in the pathogenesis of several main cardiovascular diseases including, cardiac hypertrophy, heart failure, atherosclerosis, vasodilation, revascularization and ischemic heart disease. Also, it causes vasodilatation of blood vessels and attenuates C-reactive protein-induced angiogenesis. What’s more? its ability to decrease insulin resistance in combination with its anti-atherogenic and anti-inflammatory properties makes it a hopeful therapeutic target [2]. Recent studies provided commentaries on specific aspects of omentin biology and function and explore its beneficial actions by opposing obesity and its negative effects. In addition to its diagnostic and prognostic values, it may act as a novel biomarker of obesity associated cardiovascular disorders.

Onur I et al. [3] evaluated whether serum omentin-1 levels are associated with peripheral artery disease and its severity. They concluded that it plays a role in atherosclerosis and may be a novel biomarker for peripheral artery disease. Wang XH et al. [4] investigated the alteration of plasma levels of omentin-1 in elderly patients with coronary heart disease and heart failure. They found decreased levels of omentin-1 which may be involved in the occurrence and development of the disease. Yildiz Set al. [5] evaluated whether serum omentin levels are associated with coronary artery disease and its severity among post-menopausal women. Their data indicated that a decreased omentin level is associated with the disease and its severity among postmenopausal women. Shibata et al. [6] found circulating omentin levels were independently and negatively associated with carotid intima-media thickness. Furthermore, Jalal et al. [7] demonstrated that omentin was positively correlated with high-density lipoprotein cholesterol, and inversely with glucose and triglycerides. In a study by Zhong et al. [8] Omentin levels were lower in patients with acute coronary syndrome or stable angina pectoris than those in controls. Patients with acute coronary syndrome also had lower serum concentrations of omentin compared with patients with stable angina pectoris. They concluded that, serum omentin might be a potential biomarker to predict the development and progression of coronary diseases in obesity. Kataoka et al. [9] demonstrated omentin’s functions as an adipokine that ameliorates acute ischemic heart injury by suppressing myocyte apoptosis. Thus, omentin may represent a novel target molecule for the prevention of ischemic heart disease. Another studies demonstrated that omentin ameliorates arterial calcification suggesting that the lower omentin levels in obese (especially visceral obese) subjects contribute to the development of arterial calcification, and omentin may play a protective role against it [10,11]. Moreover, Narumi et al. [12] found that decreased serum omentin levels predicted cardiac events in patients with heart failure. They concluded that, serum omentin level appears to be a novel prognostic marker for the risk stratification of patients with heart failure. Matsuo et al. [13] demonstrated that omentin attenuates the pathological process of myocardial hypertrophy suggesting that omentin may represent a target molecule for the treatment of cardiac hypertrophy.

With respect to the above mentioned cardiovascular biology of the omentin, it may have beneficial effects on the metabolic syndrome and could potentially be used as a biologic marker and pharmacologic mediator in this respect. The knowledge of these concepts may provide a new strategy to reduce disease risks on cardiovascular diseases in the future [14].
Conclusion

The potential diagnostic, prognostic, and therapeutic roles of omentin for the cardiovascular complications of obesity and the identification of omentin's receptors will be challenging topics in the future. Consequently, future therapeutic approaches to increase circulating omentin level which could be beneficial for prevention of cardiovascular diseases, necessitate to be further investigated. Much work remains to be done to interpret the fascinating biology of omentin, which will likely benefit the obese individuals with or without heart diseases.

References